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Environmental Factors Influencing Growth and Pubertal Development

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Postnatal growth is based on hereditary signals and environmental factors in a complex regulatory network. Each factor must be in an optimal state for normal growth of the child. Fetal conditions may also have consequences on postnatal height. Intrauterine growth retardation can be recovered postnatally, although postnatal growth remains depressed in about one-third of cases. After birth, the environment may exert either a positive or negative effect on growth. In underdeveloped countries, malnutrition plays a major role in inhibiting the growth process. Children from families of higher socioeconomic classes are taller than their coevals in the lower socioeconomic groups. Urbanization also has a positive effect on growth. Better child care is supported by sufficient food supply, appropriate health and sanitation services, and a higher level of education. Over the last century, these factors have induced a taller stature and a more rapid maturity in Europe, North America, and Australia; a phenomenon which has been referred to as "the secular trend" in growth. Recently, a secular trend has also been reported in some developing countries. Although urbanization in general appears to be associated with better conditions of living, this is not the case in the slums of South America or in Africa where rural children are better off than children living in the poor cities. This paper describes in more detail the different hereditary and environmental factors that act during the fetal period and postnatally, and which play a role in human growth and pubertal development.

Introduction

Growth is the result of the concerted effect of a complex network of many regulatory factors with varying interactions. Each individual has a genetic base with a definite growth potential, which may be modulated by these factors both in the prenatal period and in postnatal life (*1*). Optimal growth can only be achieved when all these factors operate in harmony.

Postnatal growth is determined by hereditary factors, the length of the newborn baby which was achieved prenatally, and environmental factors to which the child is exposed during the growth period postnatally (Fig. 1). During puberty, the pubertal growth spurt produces an extra increase in height, but thereafter growth soon ends (*2*). There is a close relationship between pubertal development and the growth process, and the onset of puberty is more correlated with skeletal age than with chronological age (*3*). Therefore, when growth is retarded, there will usually be an associated retardation of skeletal maturation, and this will result in delayed puberty as an additional complication. In many countries, the environmental conditions are such that there is incomplete expression of hereditary components, and this may have consequences on prenatal and postnatal growth.

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Genetic Factors

It is well known that the parents' height has an influence on the stature of their children. However, the relationship between the height of the baby and that of the parents is not apparent at birth but becomes more evident toward the age of 2 years, and thereafter the correlation becomes greater with increasing age (*4*). The Louisville Twin Study examined height data longitudinally from birth to maturity in twin families, and from this it was estimated that heredity accounted for 90% or more of the factors that determined height from the age of 6 years and after (*5*). These investigators observed a substantial and constant correlation between the height of the children and their parents from the age of 3 years and onwards. Monozygotic twins, with identical genetic composition, had a greater difference in final height when reared apart than when reared together. However, this difference was less than the difference between dizygotic twins (*6*). The difference in height of monozygotic twins is probably caused by environmental factors.

Body proportions are probably also under the influence of genetic control. In relative terms, the Australian Aborigines and the Africans in Ibadan have the longest legs (*1*). During the growth phase, the gain of leg length in comparison to gain in total length can be proportionally

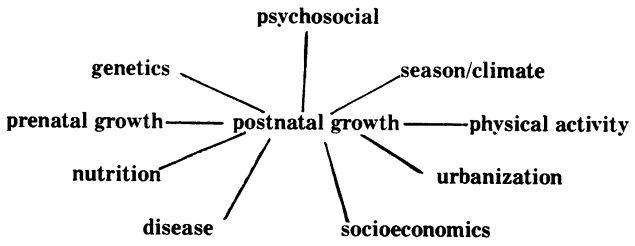

FIGURE 1. Factors influencing postnatal growth.

different in different populations. As an example, Chinese children at a young age have relatively long legs, but as they become taller they gain less leg length per unit length of sitting height than London children. The mixing of races produces children with stature and body proportions intermediate between the parental populations (*1*).

Hereditary diseases and chromosomal aberrations may affect the growth process, usually exerting a suppressive influence. Turner's syndrome (karyotype 45,XO), other X chromosomal abnormalities, and Klinefelter's syndrome are well-known diseases associated with either short or long stature. In spite of many new developments in endocrine therapy such as growth hormone treatment for Turner's syndrome, it is difficult to manipulate genetically defined growth characteristics (*7*).

Environmental Factors

Prenatal Growth

The fetus does not develop optimally in poor environmental conditions. Weight gain is the first parameter to be inhibited, but after prolonged inadequacy height is also negatively affected (*8*). Environmental conditions account for about 60% of the variability of birth weight and genetic factors for the remaining 40% (*9*). Such environmental factors, among others, include maternal age, order of birth, and crowding within the uterus (*10*). Primiparous mothers either older than 38 or younger than 20 years of age have an increased risk of giving birth to small-for-date babies (*11*). First-born babies have a birth weight of about 100 g less than second or third babies, and in multiple pregnancies the weight gain of each fetus after the 30th week of gestation is less than that of single-pregnancy fetuses (*12*). An inhibiting effect on fetal growth is also exerted by illness in the mother, malnutrition, therapeutic drug treatment, alcohol and other social drug addiction, and cigarette smoking.

Offspring of mothers with insulin-dependent diabetes are known to be at greater risk of developing congenital malformations, and the incidence of abnormalities is related to poor control of blood sugar levels in the first trimester. It is important to ensure precise diabetic control early in gestation so that a more normal environment of glucose, insulin, and ketone levels is maintained in order to diminish congenital anomalies (*13*). Antihypertensive and anticonvulsant drugs in particular are therapeutic agents that have a disturbing effect on fetal growth and morphogenesis (*14*).

Alcohol, drug addiction, and smoking may have a severe effect on the height and weight of babies (*15–17*), and smoking is known to increase the risk of prematurity (*18*). The underlying mechanisms appear to be maternal malnutrition with a deficiency of trace elements and placental dysfunction in addition to a direct toxic effect on the fetus. Alcohol addiction also increases the incidence of congenital malformations (*19*).

Malnutrition is still a worldwide problem. Fetal growth is inhibited by maternal malnutrition whether it is a deficiency of protein, calories, or trace elements. Furthermore, malnutrition may reduce fetal brain development (20).

There are three phases of cellular growth and organ development, the first being a phase of cell proliferation, followed by a phase of proliferation with concomitant hypertrophy, and a third phase of hypertrophy alone. Disturbances of the proliferation phase of brain tissue, for example, results in a lower DNA and protein content, which is irreversible and from which the brain does not recover. Therefore, the earlier the phase during which malnutrition occurs, the more serious is the lack of brain growth. This explains why fetal malnutrition may induce long-term damage to the child. Since brain development continues postpartum into infancy it is clear that postnatal malnutrition may also affect the brain (*21*).

Climate also has a regulatory effect on birth weight. Babies born in the mountains of Peru on average weigh 1500 g less than the newborns of Lima (*22*).

The socioeconomic environment in even the well-developed countries is still undergoing changes, and modern women have the opportunity of working in male-oriented industries. Over the next decade, information will be gathered about possible factors such as toxins and workload, which may interfere with providing a safe internal environment for the developing fetus (*23*). The prenatal effects on weight and height may disappear postnatally. Catch-up growth with respect to height occurs during infancy, but this may be incomplete (*24*), and the final height may be severely compromised by prenatal factors.

Postnatal Growth

Postnatal environmental factors affecting growth include nutrition, disease, socioeconomic status, urbanization, physical activity, climate, and psychosocial deprivation.

Nutrition. Malnutrition results in failure to grow, involving both weight and height. Increased growth hormone secretion occurs in protein malnutrition, presumably inducing mobilization of the remaining fat tissue (*25*). On the other hand, growth hormone levels are decreased in calorie malnutrition. When malnutrition is corrected, the affected children soon recover, and when this reversal occurs at a young age, most children will attain a complete remission in height and weight to equal their siblings before puberty (*26,27*). However, this is not always the case, probably because of long-term deficits, and the home diet following hospital admission may play a role in such an

incomplete recovery, although the deficiency in the home diet is not known in most studies (*28,29*).

More calories are required during adolescence. Anorexia nervosa is a common disease in adolescent girls, and the lack of calories results in a reduced or delayed pubertal growth spurt. Endocrine changes may also occur depending on the severity of the anorectic state, such as increased growth hormone levels associated with suppressed gonadotropin and sex steroid levels (*30*). These are mainly the consequences of malnutrition, but a central mechanism with direct effects on hypothalamic function may also be involved (*31*). Synthesis of neurotransmitters is dependent on the availability of precursors, and these may be affected by change of diet. A change in the neurotransmitters modulating hypothalamic and pituitary secretions may therefore be partially responsible for the endocrine changes associated with anorexia nervosa.

Skeletal development is essential for the growth process, and the different hormones involved in growth each have their own regulatory effect on skeletal maturation. Malnutrition causes retardation of skeletal malformation. When there are periods of arrested growth caused by either disease or malnutrition, demarcation lines can be seen on X-ray films (*32*).

Disease. The growth process is inhibited by chronic diseases, and in cases of unexplained growth failure, long-term observation may reveal underlying chronic conditions such as "asymptomatic" coeliac disease (*33*). Total recovery from chronic disease allows catch-up growth to occur, and this has two components: the first is a rapidly achieved complete restoration of growth rate to normal values, and the second is a slow recovery of growth rate to increased values, which is not as marked as the first component but is more prolonged (*34*). An alternative outcome is an extended growing period allowing normal final height to be reached but at a later time (*35*).

Socioeconomic Status. A higher socioeconomic status includes higher income associated with better education, resulting in better nutrition, better child care, and better medical and social services. These factors may induce a change in size, rate of growth, and timing of pubertal development, the so-called secular change (*1*). In Europe during the last 100 years, people have become taller, the onset of puberty has commenced at a younger age, and the full-grown state has been reached at an earlier age. However, during situations of deprivation, a decrease in growth can be detected (*37*).

The British National Child Development Survey showed a height difference of 3.3 cm in 7-year-old boys from professional and managerial classes compared to the unskilled manual working class (*36*), and this gap still remains in spite of the secular trend. Developing countries also show taller stature and greater weight attained by children from the higher socioeconomic classes (*1*).

The prevalence of overfeeding is much higher in the lower social classes in highly industrialized countries, especially where the food supply is adequate but the diet is not. In these instances, obesity is the result of poor nutritional balance with a high intake of carbohydrates and added sugars (*38*). In contrast, in developing countries the opposite is the case: the higher the income level, the higher the prevalence of obesity (*39*).

Urbanization. It is thought that urbanization results in a taller stature (*40*), and this is probably a result of sufficient food supply, adequate health and sanitation services, education, recreation, and welfare. Such a positive development is found in Europe, the United States, and Australia, but obviously not in the slums of South America or in Africa. When the state of the economy is very high as in the United States and Australia, the urban–rural difference in stature disappears (*41,42*). In developing countries where the slums are overcrowded with very poor people, urban slum children attain heights and weights similar to rural children (*43*).

Industrialization brings the problems of waste disposal sites. In 1987 Paigen et al. (*44*) described the growth pattern of children living near Love Canal in Niagara Falls in the State of New York. This is an unfinished canal 3,000 m in length, which was used as a burial site for 19,000 metric tons of organic solvents, chlorinated hydrocarbons, acids, and other hazardous wastes during the 1940s. The children under investigation who were living in this area had a shorter stature for their age than control children.

Psychosocial Stress. Acute stress elicits a burst of growth hormone secretion, but chronic exposure to stress such as is caused by psychosocial deprivation suppresses growth hormone secretion, resulting in failure to grow (*45*). Once the stress is removed, growth hormone secretion rapidly returns to normal and then there is a period of catch-up growth. This effect presumably explains the phenomenon previously described by Friend and Bransby in 1947 (*46*), who reported slow growth of children while staying at boarding school and increased growth rate during the holidays.

Season and Climate. During the year, there are periods of more rapid growth when growth rate is three times greater than the time of slowest growth. These periods of growth rate are synchronized with the seasons, and most rapid growth occurs in spring (*47*). In the tropics, lower food supply during the rainy season may be responsible for the changes in growth rate. Climate is also affected by high altitude, where people exposed to a lower oxygen saturation in the air have a shorter stature (*1*). Seasonal food scarcities may be one of the causative factors (*48*).

An investigation in Peru showed that taller stature does not necessarily imply a reproductive advantage. In industrialized countries, taller women in general have a more successful reproductive outcome than shorter women (*49*), but in the harsh environment of the Peruvian Andes, short women are more likely to produce surviving offspring. Body size, therefore, appears to be more adaptive under different ecological conditions (*50*).

Physical Activity. There are many contradictory reports about the effects of physical activity on height, puberty, and skeletal maturation (*10*), and a clear positive or negative effect on final height has never been confirmed. The effect on puberty may depend on the type of physical activity. A retarded menarche is found in girls actively involved in running or dancing, whereas there is a ten-

dency toward early maturation among swimmers. However, self-selection may be the basis of this difference. If the sport itself does have an effect on body development, it may be limited to those who indulge in intensive training.

Puberty

The onset of puberty, in addition to the rate of maturation, appears to be dependent on heredity and the environment. The age at menarche of identical twin sisters is within 1–2 months of each other, whereas in dizygotic twins there is about a year's difference (*51*). In general, girls in poor countries have later menarche than in countries with better socioeconomic conditions (*1*).

The secular trend over the last century includes a change in the timing of puberty and also a change in the rate of maturation during puberty. Environmental factors have an important bearing on this trend, and the secular changes affecting height and puberty are not always associated. For example, in the period from 1965 to 1980, women in the Netherlands became taller, whereas the age at menarche remained almost constant (*52*). In the last 20 years, the secular trend is slowing down in Europe (*53*).

The earlier age at menarche in Southern Europe may be due either to genetic factors or to the climate. There is more and more evidence suggesting that age at menarche is under the influence of genetic control. Australian girls in Sydney born to immigrant parents from Northwest and Central Europe have a menarcheal age of 13.1 years, whereas those born to immigrant parents from Southern Europe have menarche at 12.5 years, and both these ages are close to the menarcheal age of the parts of Europe from where the parents emigrated (*42*). The menarcheal age of American girls living in the hot and humid climate of Rio de Janeiro is not different from the girls living in the temperate United States. Overall, the assumption that climate has a major role in the timing of the onset of puberty appears to be erroneous (*54*).

Liestol (*55*) suggested that there is an important influence of social factors at a young age on the timing of puberty. He observed a clear relationship between social conditions during infancy and age of menarche in Norway, however, social conditions became less important later in childhood.

Precocious Puberty

In general, premature pubertal development is defined as the onset of sex characteristics at an age more than two standard deviations below the mean for that population. This corresponds to an age of younger than about 8 in girls and younger than about 9 in boys. When pubertal development is caused by premature activation of the hypothalamic-pituitary-gonadal axis, this is called central precocious puberty (*56*). Development of sex characteristics may also be stimulated by gonadotropin-independent sex steroid production or by intake of exogenous sex steroids, and this is pseudoprecocious puberty. Recently, Pasquino et al. (*57*) described a transient form of central precocious puberty in which regression of pubertal signs and suppression of hormone secretion occurs after a period of months of central endocrine stimulation. A slowly progressive variant of central precocious puberty has also been reported (*58*). Other forms of pubertal development are isolated breast development (premature thelarche) and isolated appearance of pubic hair (premature pubarche).

The exact incidence of central precocious puberty and the other forms of premature maturation is unknown. Kaplan and Grumbach (*59*) reported the age distribution of central precocious puberty in 96 girls using the age of onset (younger than 8 years) as the diagnostic criterion. Physical signs became apparent between the ages of 6 and 7 years in 54% of cases, between 2 and 6 years in 28%, and before 2 years of age in 18%. The age distribution for 10 boys with central precocious puberty (onset before the age of 9 years) showed 40% with pubertal signs before the age of 6 years. These authors consider that the high incidence in girls between the ages of 6 and 7 years is a reflection of the physiological variability in the normal age of puberty.

Boneh et al. (*60*) reported an increase in the incidence of isolated premature thelarche and also of central precocious puberty over the last 10 years in girls in Jerusalem. There was a significantly higher incidence in spring than in the other seasons. There have also been reports of premature thelarche in Italy and Puerto Rico with a suggestion that this has been caused by the ingestion of estrogens (*61,62*). These conclusions have not been substantiated. Over the last 20 years, European families have been increasingly adopting children from developing countries, and these children have been exposed to poor socioeconomic conditions at a young age before adoption. They should be expected to show catch-up growth and should therefore attain a taller final stature compared to the children of their native country. However, there is evidence that adopted children achieved premature puberty, which had negative influence on final height (*63,64*). A group of 107 Indian girls adopted by Swedes had a significantly earlier menarcheal age (mean 11.5 years) compared to girls in India (mean 12.8 years), although the final height was the same (*63*).

Oostdijk et al. (*64*) investigated 465 girls and 394 boys from South Korea, Colombia, India, and Indonesia adopted by Dutch families. The mean age at adoption was 2.9 ± 2.1 years. The mean age of the girls at menarche was significantly lower than the girls in their country of origin. A taller stature was expected because of catch-up growth, but the mean final height for both boys and girls was similar to the height of adults in their own countries.

It is concluded that adopted children are exposed to better conditions, which result in greater growth and an earlier puberty. These two effects counteract each other, with the earlier puberty preventing the more rapid growth from producing a greater final height. The regulatory mechanisms, which are probably central in origin, underlying the interactions between growth and maturation are unknown.

Conclusion

The final height of an individual depends on genetic factors and environmental factors and is influenced by

prenatal as well as postnatal growth. Before the age of 2 years, there is no correlation between the height of the parents and the height of the offspring, but there is a direct correlation after the age of 2.

Prenatal growth factors include maternal age, parity, alcohol consumption, drug addiction, smoking, therapeutic medication, climate, altitude, and malnutrition. Prenatal malnutrition has an effect on the developing brain, and the final effects produced depend on the age at which the malnutrition occurs.

Postnatal growth is affected by nutrition, socioeconomic factors, disease, urbanization, psychosocial stress, and physical activity. There is a complex interaction among these different factors, and periods of retardation can be compensated by ensuing catch-up growth if the adverse factors are remedied. Final height is determined by an interaction of growth rate and age at puberty, however, optimal conditions that stimulate growth may also advance the age of puberty with a negligible net effect on adult height.

REFERENCES

1. Eveleth, P. B., and Tanner, J. M. Worldwide Variation in Human Growth. Cambridge University Press, Cambridge, 1990.
2. Bourguignon, J. P. Linear growth as a function of age at onset of puberty and sex steroid dosage, therapeutic implications. Endocrinol. Rev. 9: 467–488 (1988).
3. Marshall, W. A., and de Limongy, Y. Skeletal maturity and prediction of age at menarche. Ann. Hum. Biol. 3: 235–245 (1976).
4. Smith, D. W., Truog, W., and Rogers, J. E. Shifting linear growth during infancy: illustration of genetic factors in growth from fetal life through infancy. J. Pediatr. 89: 225–230 (1976).
5. Philips, K., and Matheny, A. P. Quantitative genetic analysis of longitudinal trends in height: preliminary results from the Louisville Twin Study. Acta Genet. Med. Gemellol. 39: 143–163 (1990).
6. Shields, J. Monozygotic Twins. Oxford University Press, London, 1962.
7. Ranke, M. B., and Rosenfeld, R. Turner's Syndrome and Growth Promoting Therapies. Excerpta Medica, Amsterdam, 1991.
8. Usher, R. H., and McLean, F. H. Normal fetal growth and the significance of fetal growth retardation. In: Scientific Foundations of Paediatrics (J. A. Davis, J. Dobbing, and W. Heinemunn, Eds.), Medical Books Ltd., London, 1974, pp. 69–79.
9. Polani, P. E. Size of Birth. Ciba Foundation Symposium, Exerpta Medica, Amsterdam, 1974.
10. Giovannelli, G., Bernasconi, S., and Ghizzoni, L. Environmental factors and growth. In: Growth Abnormalities (J. R. Bierich, E. Cacciarci, and S. Raiti, Eds.), (Serono Symposia Publications, Vol. 56) Raven Press, New York, 1989.
11. Lobl, M., Welcher, D. W., and Mellits, E. D. Maternal age and intellectual functioning of offspring. Johns Hopkins Med. J. 128: 347–361 (1971).
12. Underwood, L. E. In: Textbook of Endocrinology (J. P. Wilson and D. W. Foster, Eds.), Saunders Co., Philadelphia, 1985.
13. Miodovnik, M., Mimouni, F., Dignan, P. S. J., Berk, M. A., Ballard, J. L., Siddiqi, T. A., Khoury, J., and Tsang, R. C. Major malformations in infants of IDDM women. Diabetes Care 11: 713–718 (1988).
14. Hill, R. M., and Tennyson, L. M. Maternal drug therapy: effect on fetal and neonatal growth and neurobehavior. Neurotoxicology 7: 121–140 (1986).
15. Jones, K. L., Smith, D. W., Ulleland, C. N., and Streissguth, A. P. Pattern of malformation of chronic alcoholic mothers. Lancet i: 1267–1268 (1973).
16. Fulroth, R. Philips, B., and Durand, D. J. Perinatal outcome of infants exposed to cocaine and/or heroin in utero. Am. J. Dis. Child. 143: 905–910 (1989).
17. Hoff, C., Wertelecki, W., Blackburn, W. R., Mendenhall, H., Wiseman, H., and Stumpe, A. Trend associations of smoking with maternal, fetal and neonatal morbidity. Obstet. Gynecol. 68: 317–321 (1986).
18. Fedrick, J., and Adelstein, P. Factors associated with low birth weight of infants delivered at term. Br. J. Obstet. Gynecol. 85: 1–7 (1978).
19. Streissguth, A. P., Heiman, C. S., and Smith, D. W. Intelligence, behaviour and dysmorphogenesis in the fetal alcohol syndrome: a report on 20 patients. J. Pediatr. 92: 363–367 (1978).
20. Winick, M. Cellular growth during early malnutrition. Pediatrics 47: 969–978 (1971).
21. Winick, M., and Rosso, P. The effect of severe early malnutrition on cellular growth of human brain. Pediatr. Res. 3 : 181–184 (1969).
22. Krugher, H., and Arias-Stella, J. The placenta and the newborn infant at high altitudes. Am. J. Obstet. Gynecol. 106: 586–591 (1970).
23. McCloy, E. C. Work, environment and the fetus. Midwifery 5: 53–62 (1989).
24. Fitzhardinge, P. M., Inwood, S. Long-term growth in small-for-date children. Acta Paediatr. Scand. (suppl.) 349: 27–33 (1989).
25. Primestone, B. L., Barbezat, G., Hansen, J. D. L., and Murray, P. Studies on growth hormone secretion in protein-calorie malnutrition. Am. J. Clin. Nutr. 21: 482 (1968).
26. Garrow, J. S., and Pike, M. C. The long-term prognosis of severe infantile malnutrition. Lancet i: 1–4 (1967).
27. Hansen, J. D. L., Freesemann, C., Moodie, A. D., and Evans, D. E. What does nutritional growth retardation imply? Pediatrics 47: 299–313 (1971).
28. MacWilliam, K. M., and Dean, R. F. A. The growth of malnourished children after hospital treatment. East African Med. J. 42: 297–304 (1965).
29. Graham, G. G. The later growth of malnourished infants: effects of age, severity and subsequent diet. In: Calorie Deficiencies and Protein Deficiencies (R. A. McCance and E. M. Widdowson, Ed.), Churchill, London, 1968, pp. 301–316.
30. Brown, G. M., Seggie, J. A., Chambers, J. W., and Ettigi, P. G. Psychoneuro-endocrinology and growth hormone: a review. Psychoneuroendocrinology 3: 131–153 (1978).
31. Eisenberg, E. Toward an understanding of reproduction function in anorexia nervosa. Fertil. Steril. 36: 543–550 (1981).
32. Blanco, R. A., Acheson, R. M., Canosa, C., and Salomon, J. B. Height, weight and lines of arrested growth in young Guatemalan children. Am. J. Physical. Anthropol. 40: 39–48 (1974).
33. Groll, A., Candy, D., Preece, M., Tanner, J., and Harries, J. Short stature as the primary manifestation of coeliac disease. Lancet ii: 1097–1099 (1980).
34. Prader, A., Tanner, J. M., and von Harnack, G. A. Catch-up growth following illness or starvation. J. Pediatrics 62: 646–659 (1963).
35. Tanner, J. M. Catch-up growth in man. Br. Med. Bull. 37: 233–238 (1981).
36. Goldstein, H. Factors influencing the height of seven-year-old children. Results from the National Child Development Study. Hum. Biol. 43: 92–111 (1971).
37. Douglas, J. W. B., and Simpson, H. R. Height in relation to puberty, family size and social class. Millbank Mem. Fund Q. 42: 20–35 (1964).
38. Brindtland, G., Liestol, K., and Walloe, L. W. Height, weight and menarcheal age of Oslo school children during the last 60 years. Ann. Hum. Biol. 7: 307–322 (1980).
39. Cook, J., Altman, D. G., Morre, D. M. C., Topp, S. G., Holland, W. W., and Elliot, A. A survey of the nutritional status of schoolchildren. Relation between nutrient intake and socio-economic factors. Br. J. Prevent. Soc. Med. 27: 91–99 (1973).
40. Tanner, J. M., and Eveleth, P. B. Urbanisation and growth. In: Man in Urban Environments (G. A. Harrison and J. B. Gibson, Eds.), Clarendon Press, Oxford, 1976, pp. 144–166.
41. Hamill, P. V. V., Johnston, F. E., and Lemeshow, S. Height and Weight of Children: Socio-Economic Status. Department of Health, Education and Welfare Publication, No. 73-1606, Vital Health Statistics Series 11, No. 119. U.S. Goverment Printing Office, Washington, DC, 1972.
42. Jones, D. L., Hemphill, W., and Meyers E. S. A. Height, weight and other physical characteristics of New South Wales children. Part 1. Children aged five years and over. New South Wales Department of Health, G.96543-a K5705, Sydney, Australia, 1973.
43. Villarejos, V. M., Osborne, J. A., Payne, F. J., and Arguedes, J. A. Heights and weights of children in urban and rural Costa Rica.

Environ. Child Health 17: 31–43 (1971).
44. Paigen, B., Goldman, L. R., Magnant, M. M., Highland, J. H., and Steegmann, A. T. Growth of children living near the hazardous waste site, Love Canal. Hum. Biol. 59: 489–508 (1987).
45. Kriger, I., and Mellinger, R. C. Pituitary function in the deprivation syndrome. J. Pediatr. 79: 216–225 (1971).
46. Friend, G. E., and Bransby, E. R. Physique and growth of schoolboys. Lancet ii: 677–681 (1947).
47. Marshall, W. A. Evaluation of growth rate in height over periods of less than one year. Arch. Dis. Child. 46 :414–420 (1971).
48. Leonard, W. R. Nutritional determinants of high altitude growth in Nunoa, Peru. Am. J. Physical. Anthropol. 80: 341–352 (1989).
49. Thomson, A. M. Maternal stature and reproductive efficiency. Eugenics Rev. 51: 157–162 (1954).
50. Frisancho, A. R., Sanchez, J., Pollardel, D., and Yanez, L. Adaptive significance of small body size under poor socioeconomic conditions in Southern Peru. Am. J. Physical Anthropol. 39: 255–262 (1973).
51. Fischbein, S. Intra-pair similarity in physical growth in monozygotic and of dizygotic twins during puberty. Ann. Hum. Biol. 3: 205–210 (1977).
52. Roede, M. J., and Wieringen, J. C. Growth diagrams 1980. T. Soc. Gezondheidszorg 63 (suppl.): 1–34 (1985).
53. Danker-Hopfe, H. Die Säkulare Veranderung des Menarchealters in Europa. Schweizerbart, Stuttgart, 1984.
54. Eveleth, P. B. Eruption of permanent dentition and menarche of American children living in the tropics. Human Biol. 38: 60–70 (1966).
55. Liestol, K. Social conditions and menarcheal age: the importance of early years of life. Ann. Hum. Biol. 9: 521–538 (1982).
56. Stanhope, R., and Brook, C. G. D. In: Clinical Paediatric Endocrinology: Disorders of Puberty (C. G. D. Brook, Ed.), Blackwell, Oxford, 1989, pp. 189–212.
57. Pasquino, A. M., Cives, C., Maciocci, M., Tebaldi, L., Musleh, M., and Boscherini, B. Transient true precocious puberty. A report of five cases. Eur. J. Pediatr. 148: 735–736 (1989).
58. Fontoura, M., Brauner, R., Prevot, C., and Rappaport, R. Precocious puberty in girls: early diagnosis of a slowly progressing variant. Arch. Dis. Child. 64: 1170–1176 (1989).
59. Kaplan, S. L., and Grumbach M. M. Pathogenesis of sexual precocity. In: Control of the Onset of Puberty, II (M. M. Grumbach, P. C. Sizonanko, and M. L. Aubert, Eds.), Williams and Wilkins, Baltimore, MD, 1990, pp. 620–664.
60. Boneh, A., Landau, H., and Friedlander, N. Age and seasonal factors in the incidence of premature sexual development in girls in the Jerusalem area. Clin. Invest. Med. 12: 172–174 (1989).
61. Fara, G. M., Del Corvo, G., Bernuzzi, S., Bigatello, A., Di Pietro, C., Scaglioni, S., and Chiumello, G. Epidemic of breast enlargement in an Italian school. Lancet ii: 295–297 (1979).
62. Bongiovanni, S. M. An epidemic of premature thelarche in Puerto Rico. J. Pediatr. 103: 245–246 (1983).
63. Proos, L. A., Holvander, Y., and Tuvvemo, T. Height and weight at menarche for Indian girls adopted to Sweden (abstract no. 4). In: Third International Conference on the Control of the Onset of Puberty, Amsterdam, 1989.
64. Oostdijk, W., Yap, Y. N., Doude van Troostwijk, M. L., Gevers, E. F., Brand, R., Verhulst, F. C., and Drop, S. L. S. Final height and timing of puberty of adopted children in the Netherlands. Hormone Res. 35(2): 24 (1991).